

detected. Compared with the controls, the differential expressions of 47 mRNAs had statistical significance ( $P < 0.05$ ). 3) In PE patients, multiple linear regression analysis indicated that the expressions of CXCL3 mRNA had low positive correlations with ITGA2B ( $R^2 = 0.247$ ) and ITGB3 ( $R^2 = 0.247$ ); The CXCL16 mRNA expression had a low positive correlation with ITGA2B ( $R^2 = 0.201$ ); The CXCL14 mRNA expression had a low negative correlation with ITGB3 ( $R^2 = 0.212$ ).

**Conclusions:** The results suggest that, in symptomatic PE patients, CXCL3, CXCL14 and CXCL16 mRNA were the cytokines which related to the activation of platelet. CXCL3, CXCL14 and CXCL16 could be the triggering factors for the activation and aggregation of platelets.

#### GW25-e0503

##### The congenital and acquired thrombophilia in Chinese pulmonary thromboembolism patients

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**Objectives:** The genetic polymorphisms of coagulation factor V Leiden and prothrombin G20210A are common risk factors of inherited thrombophilia in Caucasians, but rare in Aisan. Factor V is an substrate of the activated protein C anticoagulant system. While in Asian population, dysfunction of APC system including protein C and protein S, and antithrombin is involved in thrombophilia. The coagulation inhibitor protein deficiency consists of Protein C (PC), protein S (PS) and antithrombin (AT). The previous studies showed that the prevalence of coagulation inhibitor protein deficiency was 7% in European (Heijboer H, et al), 65% in Japanese (Kinoshita S, et al), 48% in Hong Kong Chinese (Liu HW, et al), 59% in Taiwan Chinese (Shen MC, et al) and 29% in mainland Chinese (Bai CM, et al). It seems to be low estimated in mainland Chinese. As for acquired thrombophilia, antiphospholipid syndrome and hyperhomocysteinemia were the common reasons.

**Methods:** From Jan 2011 to Dec 2013, consecutive 139 patients (71 male) of pulmonary thromboembolism in the First Hospital of Jilin University were enrolled in this study with exclusion of underlying diseases such as malignancy, myeloproliferative disorder, nephrotic syndrome, liver disease and oral warfarin. The mean age was  $58 \pm 14$  yrs. The diagnosis was confirmed by CT pulmonary angiography. Before oral warfarin taken, the activity of blood protein C, protein S and antithrombin, as well as serum anticardiolipin antibody (aCL) and homocysteinemia (Hcy) was assayed. The normal range of the factors above is PC 70-140%, PS 76-135%, AT 80-120%, aCL (IgG and IgM) 0-10U/L, Hcy 0-20  $\mu$ mol/L. The prevalence of kinds of thrombophilia factor was calculated as the ratio of summary number of isolated and combined containing the defined factor. The combined deficiency included PC+PS, PC+AT, PS+AT or PC+PS+AT. The prevalence of coagulation inhibitor protein deficiency means the ratio of total number of patients with all kinds isolated or combined coagulation inhibitor protein deficiency.

**Results:** The case number and prevalence of kinds of thrombophilia factor is PC 32 [23.0% (95%CI:16.0%-30.0%)], PS 65 [46.7% (38.5%-55.1%)], AT 14 [10.1% (5.1%-15.1%)], aCL 14 [10.1% (5.1%-15.1%)], Hcy 30 [21.6% (14.7%-28.4%)], combined deficiency 25 [18.0% (11.6%-24.4%)]. The number of isolated deficiency cases is PC 10, PS 42 and AT 5. The combined deficiency includes PC+PS 16, PC+AT 2, PS+AT 3, PC+PS+AT 4 respectively. The number of isolated acquired thrombophilia factor is aCL 3 and Hcy 12. There are also one combined acquired thrombophilia (aCL+Hcy) case and combined congenital and acquired cases (PC+Hcy 3; PS+aCL 8; PS+Hcy 7; AT+aCL 1; AT+Hcy 1; PC+PS+aCL 1; PC+PS+Hcy 5; PC+PS+AT+Hcy 1). The total number and prevalence ratio of patients with any kinds of congenital or acquired thrombophilia factor is 98 (70.5%).

**Conclusions:** The prevalence of coagulation inhibitor protein (PC, PS and AT) deficiency in mainland Chinese is similar with that of other Aisan population. Besides the congenital factors, acquired thrombophilia such antiphospholipid syndrome and hyperhomocysteinemia should be considered in venous thromboembolism (VTE) patients. The congenital along with acquired thrombophilia factors might explain nearly 2/3 VTE occurrence.

#### GW25-e0152

##### Effect of Th17 and Treg Axis Disorder on Outcomes of Pulmonary Arterial Hypertension in Connective Tissue Diseases

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**Objectives:** This prospective cohort study is to verify the hypothesis that the balance of Th17 and Treg cells frequencies in the peripheral circulation is disturbed in patients with varying degrees of connective tissue diseases-associated pulmonary arterial hypertension (CTD-aPAH), and to prove the influence of Th17/Treg imbalance on prognosis.

**Methods:** We detected the frequencies of Th17 and Treg cells and related serum cytokines secretion and expressions of key transcription factors in 117 patients with connective tissue diseases (CTD), 53 patients with CTD-aPAH and 48 healthy volunteers. Moreover, the median value according to levels of Th17/Treg ratios in patients with CTD-aPAH was chosen as basis of group division for survival analysis.

**Results:** CTD-aPAH patients revealed significant increase in peripheral Th17 cells number, Th17-related cytokines, and ROR  $\gamma$ t mRNA levels. They also presented a significant decrease in Treg cells number, Treg-related cytokines, and Foxp3 mRNA levels as compared with CTD patients and healthy controls. More importantly, the Th17/Treg ratio was significantly related to the severity and prognosis of CTD-aPAH. **Conclusions:** This study indicated that the Th17/Treg axis disorder play a critical role in CTD-aPAH. Furthermore, the dynamic balance between Th17 and Treg cells was likely to influence prognosis of patients with CTD-aPAH.

#### GW25-e0259

##### The Expression of Fibrinogen-Related Domain Associated Genes mRNA in Patients with Symptomatic Pulmonary Embolism

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**Objectives:** To investigate the mRNA expression characterizations of FGA, FGB, FGG, FIBCD1 and FCN, which were associated with fibrinogen-related domain, between patients with symptomatic pulmonary embolism and controls in their peripheral blood mononuclear cells.

**Methods:** 20 cases of PE patients and twenty sex and age matched controls were recruited into the study. Human cDNA microarray analysis was used to detect the mRNA expression differences of FGA, FGB, FGG, FIBCD1 and FCN between two groups, and random variance model corrected t test was used to analyze the statistical data.

**Results:** Among the 7 mRNAs, the mRNA expressions of FGA, FGB and FGG were down-regulated significantly in the PE group, compared with the controls ( $P < 0.05$ ); FIBCD1, FCN-1 and FCN-2 were up-regulated in the PE group ( $P < 0.01$ ); FCN-3 in PE was up-regulated, but there was no significant difference ( $P > 0.05$ ).

**Conclusions:** The mRNA expression of FGA, FGB and FGG in PE indicate that body's feedback mechanism plays a role to reduce further thrombosis; FIBCD1, FCN-1 and FCN-2 in PE are up-regulated significantly, suggest the expression levels of related proteins which can activate the complement system are increased, and part of innate immune molecules enhanced their function.

#### GW25-e0260

##### Differential expression of leucocyte $\beta$ 2 integrin signal transduction associated genes in patients with symptomatic pulmonary embolism

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**Objectives:** Whole human genome oligo microarray was employed to systematically investigate the differential expression characteristics of associated mRNAs which were found in the signal transduction pathway of the  $\beta$ 2 integrin in peripheral blood mononuclear cells (PBMCs) between patients with symptomatic pulmonary embolism (PE) and controls.

**Methods:** 20 cases of PE patients and twenty sex- and age-matched controls were recruited into the study. Human cDNA microarray analysis was used to detect the differences of associated mRNAs expressions between two groups, and random variance model corrected t test was used to analyze the statistical data.

**Results:** A total of 80 associated mRNAs were detected. The mRNA expressions of chemokines, ligands, inside-out signal pathway associated proteins and outside-in signal pathway associated proteins were up-regulated significantly in the PE group, compared with the controls. In 5 subunit-associated mRNAs, the mRNA expressions of ITGAL, ITGAM, ITGAX and ITGB2, which encode for the subunits of  $\alpha$ L,  $\alpha$ M,  $\alpha$ X,  $\beta$ 2, were up-regulated in the PE group, and the differences except ITGB2 were statistically significant ( $P < 0.05$ ). The mRNA expression of ITGAD was down-regulated, but there was no significant difference ( $P > 0.05$ ). The expression of Fgr mRNA was significantly down-regulated ( $P < 0.01$ ).

**Conclusions:** Thus, in PE patients, bilateral signal transduction pathways of  $\beta$ 2 integrin in neutrophils and monocytes were activated, so innate immune enhanced.

#### GW25-e0269

##### Angiotensin II receptor antagonist attenuates cigarette smoke-induced pulmonary vascular remodeling in rats: Potential involvement of angiotensin-converting enzyme-2

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**Objectives:** Renin-angiotensin system (RAS) is known to function in the development of Pulmonary vascular remodeling. Losartan, a specific angiotensin II receptor antagonist, is a well-known antihypertensive drug with a potential role in regulating angiotensin-converting enzyme-2 (ACE2). To determine the effect of losartan on smoke-induced Pulmonary vascular remodeling and its possible mechanism.